JOURNAL OF BACTERIOLOGY, Mar. 1986, p. 890–895
0021-9193/86/030890-06$02.00/0

Vol. 165, No. 3

Acquisition of Maltose Chemotaxis in *Salmonella typhimurium* by the Introduction of the *Escherichia coli* Chemosensory Transducer Gene

TAKAFUMI MIZUNO,[1]* NORIHIRO MUTOH,[2] SHARON M. PANASENKO,[3] AND YASUO IMAE[1]

Institute of Molecular Biology, Faculty of Science, Nagoya University, Chikusa-ku, Nagoya 464, Japan[1]; Division of Biology, California Institute of Technology, Pasadena, California 91125[2]; and Chemistry Department, Pomona College, Claremont, California 91711[3]

Received 14 October 1985/Accepted 3 December 1985

***Escherichia coli* and *Salmonella typhimurium* are closely related species. However, *E. coli* cells show maltose chemotaxis but *S. typhimurium* cells do not. When an *E. coli* chemotransducer gene (tar_E), the product of which is required for both aspartate and maltose chemotaxis, was introduced by using a plasmid vector into *S. typhimurium* cells with a defect in the corresponding gene (tar_S), the transformant cells acquired the ability for both aspartate and maltose chemotaxis. In contrast, when the tar_S gene was introduced into tar_E-deficient *E. coli* cells, the transformant cells acquired aspartate chemotaxis but not maltose chemotaxis. These results indicate that the absense of maltose chemotaxis in *S. typhimurium* is a consequence of the properties of the tar_S gene product.**

Bacteria, like higher organisms, have an ability to sense various kinds of stimuli in their environment and to migrate to more suitable conditions. Chemotaxis in *Escherichia coli* and *Salmonella typhimurium* has been studied extensively at the molecular level (2, 10, 19). Chemical stimuli are sensed at first by receptors located in the periplasm, and then the ligand-bound receptors interact with their specific chemotransducers embedded in the cytoplasmic membrane so that the information is transferred from the outside to the inside of the cell. Some stimuli are known to interact directly with chemotransducers (8, 26).

E. coli and *S. typhimurium* are closely related species, and their genetic organizations for chemotaxis are quite similar. Specifically, most genes for the chemosensory transducing system (*che* genes) are interchangeable (7). Furthermore, the amino acid sequences of the chemotransducers of these two species have about 80% homology, as estimated from DNA sequencing of the genes (12, 23). In spite of such a high similarity between these two species, there are some differences in their chemotactic properties. One such difference is the chemotaxis to maltose; *E. coli* shows maltose chemotaxis but *S. typhimurium* does not (3, 10).

To exhibit maltose chemotaxis in *E. coli*, maltose-binding protein located in the periplasm at first binds maltose, and then the complex interacts with a chemotransducer, the Tar_E protein, the main function of which is as the chemoreceptor-transducer for L-aspartate (9, 14, 22). Although *S. typhimurium* cells show no maltose chemotaxis, they have in their periplasm maltose-binding protein that is need for maltose transport (18), and they also have the chemoreceptor-transducer for L-aspartate, the Tar_S protein (10). What then is the cause for the behavioral difference to maltose between these two species? Which one—maltose-binding protein, chemotransducer, or the membrane in which the transducers are embedded—is responsible for the difference?

In this report, we present evidence that the chemotransducer of *S. typhimurium* has the responsibility for the absence of maltose chemotaxis in *S. typhimurium*. After this work was completed, we learned that the maltose-binding protein of *S. typhimurium* is active for the restoration of maltose chemotaxis in maltose-binding protein-deficient *E. coli* cells (6a). Their results are not only consistent but also complementary with ours.

MATERIALS AND METHODS

Bacterial strains and plasmids. The *E. coli* strains used in this study were RP487 (che^+) (20), RP5698 (Δtsr_E1-28) (16), and MS5228 [tsr_E-*1* $\Delta(tar_E$-*tap)5201*] (24). All the strains were K-12 derivatives and had a *metF* allele. The *S. typhimurium* strains used were ST1 (che^+) (5), ST330 (tsr_S-*10*), and ST334 (tsr_S-*10* tar_S-*10*). All were derivatives of strain LT-2, and the latter two chemotransducer mutants were isolated by S. M. Panasenko and D. E. Koshland, Jr. (unpublished data). ST330 showed no chemotactic response to at least 10 mM L-serine, and ST334 showed no response to at least 10 mM each of L-serine and L-aspartate.

Plasmids used were pRK41 (tar_S^+) (23) and pNM17 (tar_E^+). Both were derivatives of pBR322 and carried an ampicillin resistance marker. pNM17 was constructed from pAK101 (11) by the introduction of the *tac* promoter (4) (N. Mutoh and M. I. Simon, unpublished data).

Chemicals. L-[*methyl*-^{3}H]methionine (12.0 Ci/mmol) and En^{3}Hance were obtained from New England Nuclear Corp. (Boston, Mass.). Chloramphenicol was purchased from P-L Biochemicals, Inc. (Milwaukee, Wis.). Synthetic L-serine was the product of ICN Pharmaceuticals, Inc. (Plainview, N.Y.). α-Methyl-DL-aspartic acid was purchased from Sigma Chemical Co. (St. Louis, Mo.). Maltose was obtained from Wako Pure Chemical Industries, Ltd. (Osaka, Japan).

Transformation. Isolation of plasmid DNA and its transformation into bacteria were carried out by standard methods (13). To transform the plasmid isolated from *E. coli* strains into *S. typhimurium* strains, the plasmids were initially passed through a restriction-deficient *S. typhimurium* strain, LB5000 (6), which was kindly supplied by L. R.

* Corresponding author.

Bullas of Loma Linda University, for the host-controlled modification.

Cell growth. Cells were grown at 35°C with shaking in tryptone broth consisting of 1% tryptone (Difco Laboratories, Detroit, Mich.) and 0.5% NaCl supplemented with 0.5% glycerol and 20 mM maltose. In the case of the plasmid-carrying strains, the broth was also supplemented with 50 μg of ampicillin per ml. At the late log phase of growth (A_{590} = 0.7), cells were harvested by centrifugation at room temperature and washed three times with wash medium consisting of 10 mM potassium phosphate buffer (pH 7) and 0.1 mM potassium EDTA.

Temporal stimulation assay. The temporal stimulation assay was carried out with free-swimming cells as described previously (16). Briefly, the cells were mixed in a test tube with an attractant, and a drop of the cell suspension was immediately placed on a glass slide. Swimming cells were observed at 25°C, and the suppression of tumbling by the addition of attractant was measured. Response time was designated as the time required for about 70% of the cells to accomplish adaptation.

E. coli MS5228 showed a quite low frequency of tumbling before stimulation. To detect the smooth swimming response to attractants, a repellent, 1 M glycerol, was added to induce tumbling in MS5228 (16). Under these conditions, the smooth swimming response was clearly detected toward an attractant, 10 mM D-ribose (16).

Capillary assay. Capillary assays were performed by the method of Adler (1) with a slight modification. The washed cells were suspended in wash medium supplemented with 10 mM sodium lactate and 10 μM L-methionine. The cell concentration was adjusted to an A_{590} of 0.005. A fraction of the cell suspension (200 μl) was sucked into the yellow tip of a Gilson Pipetman (Gilson France, Villiers Le Bel, France). The tip was removed from the pipet gently and then laid down on a dish. The attractant-containing 5-μl volume capillary (Drummond Scientific Co., Broomall, Pa.) was inserted into the cell suspension in the tip. After 45 min of incubation at 30°C, the cells that accumulated in the capillary were counted as the number of colonies on a tryptone agar plate consisting of tryptone broth and 1.2% agar.

Measurement of the methylation level of chemotransducers. The methylation levels of chemotransducers were measured by the methods of Springer et al. (25) with a minor modification as described previously (15). Briefly, the washed cells (A_{590} = 1.0) in wash medium supplemented with 10 mM sodium lactate–10 μM L-[*methyl*-^{3}H]methionine (0.4 mCi/μmol)–200 μg of chloramphenicol per ml were incubated with shaking at 30°C for about 40 min. Attractants were then added, and samples (0.5 ml) were withdrawn at intervals. After separation of the chemotransducers by sodium dodecyl sulfate-polyacrylamide gel electrophoresis, the radioactivity incorporated into the transducers was measured. To make fluorograms of the methylated chemotransducers, cells were incubated with L-[*methyl*-^{3}H] methionine (2 mCi/μmol). Other procedures were the same as described previously (17).

RESULTS

Aspartate and maltose chemotaxis in wild-type strains of *E. coli* and *S. typhimurium*. It has been reported that *E. coli* shows positive chemotaxis to both aspartate and maltose, whereas *S. typhimurium* shows aspartate chemotaxis but no maltose chemotaxis (3, 10). We confirmed the results by the temporal stimulation assay (Table 1).

TABLE 1. Behavioral response to α-methyl-DL-aspartate or maltose in various strains of *S. typhimurium* and *E. coli* by temporal stimulation assay

Strain	Genotype of:		Response time (s) of[a]:	
	Host	Plasmid	α-MeAsp[b] (0–100 μM)	Maltose (0–100 μM)
S. typhimurium				
ST1	Wild type		44	<15[c]
ST330	*tsr*$_S$-*10*		38	<15
ST334	*tsr*$_S$-*10 tar*$_S$-*10*		<15	<15
ST334(pNM17)	*tsr*$_S$-*10 tar*$_S$-*10*	*tar*$_E^+$	322	157
ST334(pRK41)	*tsr*$_S$-*10 tar*$_S$-*10*	*tar*$_S^+$	>1,800	<15
E. coli				
RP487	Wild type		190	135
RP5698	Δ*tsr*$_E$1-28		224	126
MS5228	*tsr*$_E$-*1* Δ(*tar*$_E$-*tap*)*5201*		<15	<15
MS5228(pNM17)	*tsr*$_E$-*1* Δ(*tar*$_E$-*tap*)*5201*	*tar*$_E^+$	275	170
MS5228(pRK41)	*tsr*$_E$-*1* Δ(*tar*$_E$-*tap*)*5201*	*tar*$_S^+$	>1,800	<15

[a] Response time was measured as the time required for 70% of the cells to accomplish adaptation to the stimulus.
[b] α-MeAsp, α-methyl-DL-aspartate.
[c] <15, Response was not detected.

In the case of *E. coli*, it has been shown that maltose at first binds to maltose-binding protein, and then, as the trigger of sensory transduction, the complex interacts with Tar$_E$ protein which is the chemoreceptor-transducer for aspartate. It is therefore reasonable to speculate that the absence of maltose chemotaxis in *S. typhimurium* is caused by a defect somewhere in these steps. Likely candidates are assumed to be maltose-binding protein, Tar$_S$ protein, which is the chemoreceptor-transducer for aspartate and has a high similarity with the Tar$_E$ protein of *E. coli*, or the membrane in which Tar$_S$ proteins are embedded. Because both *tar*$_E$ and *tar*$_S$ genes have been cloned on plasmid vectors (12, 23), the use of these plasmids provides a simple way to investigate the role of these chemotransducer proteins in maltose chemotaxis.

Introduction of *E. coli tar*$_E$ gene into *S. typhimurium* cells. At first, an *E. coli* chemotransducer gene, *tar*$_E$, was introduced into *S. typhimurium* cells to compare the role of Tar$_E$ proteins with that of Tar$_S$ proteins in maltose chemotaxis. The recipient *S. typhimurium* strain used was ST334, which has defects in both the chemoreceptor-transducers for aspartate and serine, namely *tar*$_S$ and *tsr*$_S$ gene products, and is, therefore, nonchemotactic to both attractants. A plasmid pNM17, which carried the *tar*$_E^+$ gene, was transferred into ST334. For control experiments, plasmid pRK41, which carried a *S. typhimurium* chemotransducer gene, the *tar*$_S^+$ gene, was also transferred into ST334.

Aspartate and maltose chemotaxis of these transformants, ST334(pNM17) and ST334(pRK41), was investigated by the capillary assay method. ST334(pNM17) showed chemotaxis not only to aspartate but also to maltose, whereas the control transformant ST334(pRK41) showed chemotaxis to aspartate but not to maltose (Fig. 1). Consistent with these results was the fact that a sudden increase in α-methyl-DL-aspartate caused smooth swimming in both transformants, but an increase in maltose caused smooth swimming only in ST334(pNM17) (Table 1). Thus, these results show that the chemotransducer Tar has the responsibility for the difference in the maltose chemotaxis between *E. coli* and *S.*

typhimurium. It is noteworthy that adaptation to α-methyl-DL-aspartate in ST334(pRK41) was quite slower than that in ST334(pNM17). This is probably due to a high copy number of pRK41, as reported previously (23). Actually, ST334(pNM17) showed a larger swarm on a tryptone soft agar plate than did ST334(pRK41) (data not shown).

It has been shown that attractant stimuli induce an increase in the methylation level of the relevant chemotransducers and the increase causes adaptation of the cells to the stimuli (25). To investigate the expression of the introduced Tar_E and Tar_S proteins in the transformants, changes in the methylation level of these proteins were measured. The selective labeling of the methyl groups in these proteins were performed by incubation of the cells with ^{3}H-methyl-labeled methionine under the condition of protein synthesis inhibition (25). Because the host strain ST334 had defects in major chemotransducers, only a slight incorporation of the radioactivity was observed in ST334 cells, and this low incorporation was not affected by the addition of any attractants (data not shown). In the case of the transformants ST334(pNM17) and ST334(pRK41), a significant incorporation of the radioactivity was observed, and the incorporation was clearly enhanced by the addition of α-methyl-DL-aspartate (Fig. 2). In contrast, the addition of maltose caused an increase of the incorporation in ST334(pNM17) but not in

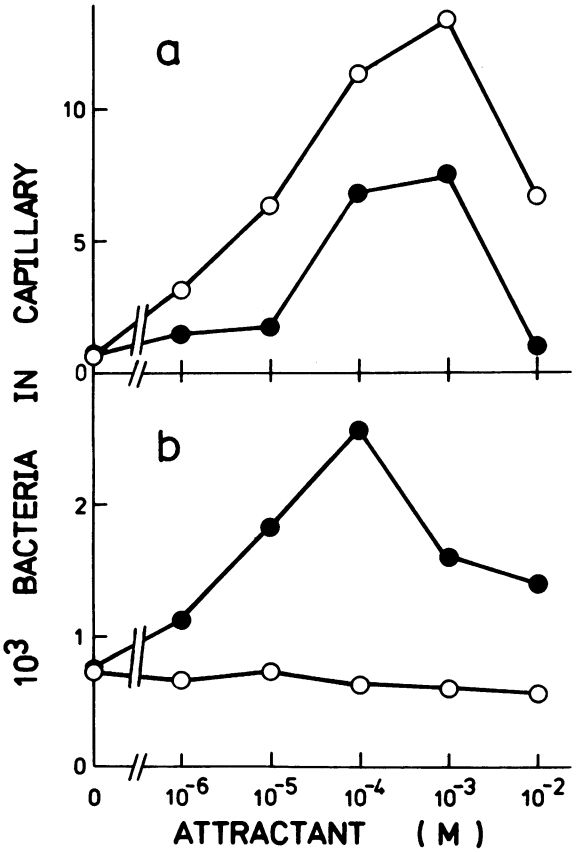

FIG. 1. Aspartate and maltose chemotaxis in *S. typhimurium* ST334 (*tar*$_S$-*10 tsr*$_S$-*10*) with a plasmid carrying the *tar*$_E^+$ (pNM17) or the *tar*$_S^+$ (pRK41) gene. Capillary assays were performed at different concentrations of α-methyl-DL-aspartate (a) or maltose (b). Symbols: ●, ST334(pNM17); ○, ST334(pRK41).

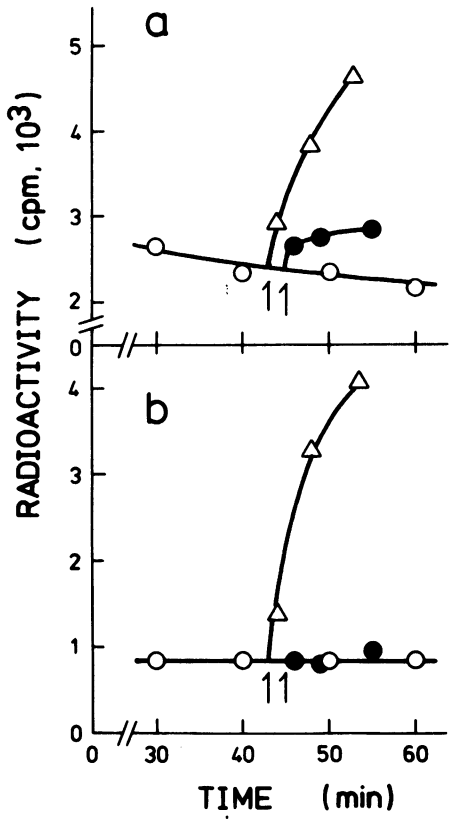

FIG. 2. Changes in the methylation level of Tar_E or Tar_S proteins in ST334(pNM17) or ST334(pRK41). Cells were incubated at 30°C with ^{3}H-methyl-labeled methionine for about 40 min and then divided into three groups. One group received distilled water (○), and the other groups received 10 mM α-methyl-DL-aspartate (△) or 10 mM maltose (●) at the time point indicated by arrows. (a) ST334(pNM17); (b) ST334(pRK41).

ST334(pRK41) (Fig. 2). These results are consistent with the behavioral results of these transformants.

Methylated chemotransducers in transformants were visualized as a fluorogram after the separation of the chemotransducers by sodium dodecyl sulfate-polyacrylamide gel electrophoresis. Tar_E and Tar_S proteins exhibited a multiple banding pattern because of their multiple methyl-accepting properties (19) (Fig. 3). The stimulation by α-methyl-DL-aspartate in both transformants caused a clear increase in the total radioactivity and in the density of faster migrating bands which are highly methylated species of the chemotransducers. Maltose was found to be effective in ST334(pNM17) but not in ST334(pRK41). Because neither transformant had a functional serine chemotransducer, serine at 10 mM had almost no effect on the incorporation of radioactivity and also on the migration pattern of the radioactivity. These results indicate that both of the introduced chemotransducers Tar_E and Tar_S are normally functioning in these transformants. It is noteworthy that there is a small but definite difference in the mobility of radioactive bands between the chemotransducers in these transformants; Tar_E proteins in ST334(pNM17) showed slightly faster migration than Tar_S proteins in ST334(pRK41). Exactly the same difference in the migration pattern was observed between Tar_E and Tar_S proteins isolated from RP5698 (Δ*tsr*$_E$1-28) and ST330 (*tsr*$_S$-*10*) (data not shown), indicating that the

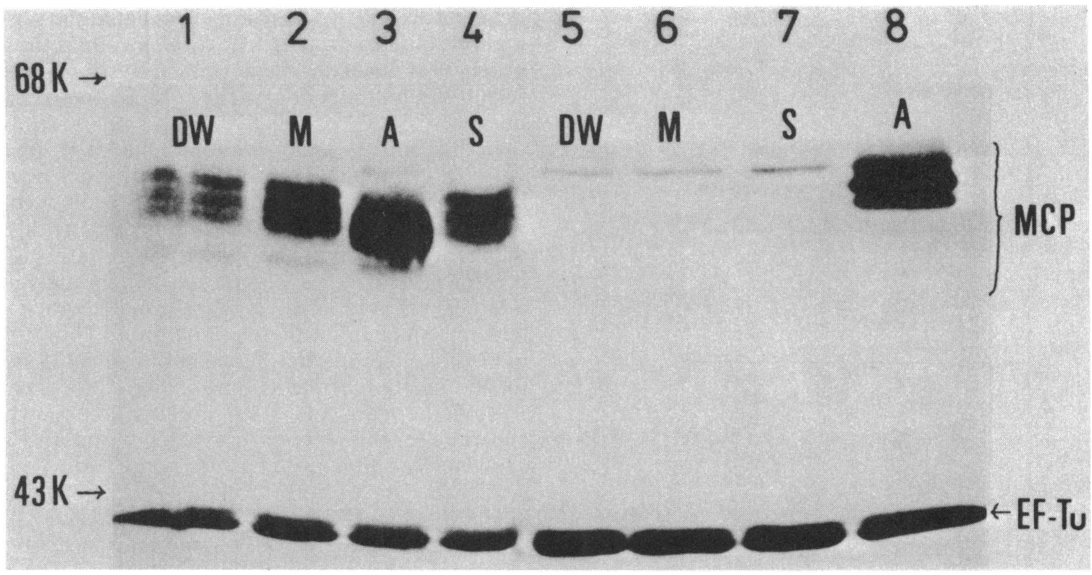

FIG. 3. Fluorogram of the methylated Tar_E or Tar_S proteins in ST334(pNM17) and ST334(pRK41). ST334(pNM17) cells (lanes 1 through 4) or ST334(pRK41) cells (lanes 5 through 8) were incubated with radioactive methionine for 40 min at 30°C. Then, distilled water or attractants were added, and the cells were incubated for 10 more min. Tar_E and Tar_S proteins were separated by sodium dodecyl sulfate-polyacrylamide gel electrophoresis, and the fluorogram was produced. Lanes 1 and 5, distilled water (DW); lanes 2 and 6, 10 mM maltose (M); lanes 3 and 8, 10 mM α-methyl-DL-aspartate (A); lanes 4 and 7, 10 mM L-serine (S). K, molecular weight (in thousands); MCP, chemotransducers; EF-Tu, elongation factor Tu.

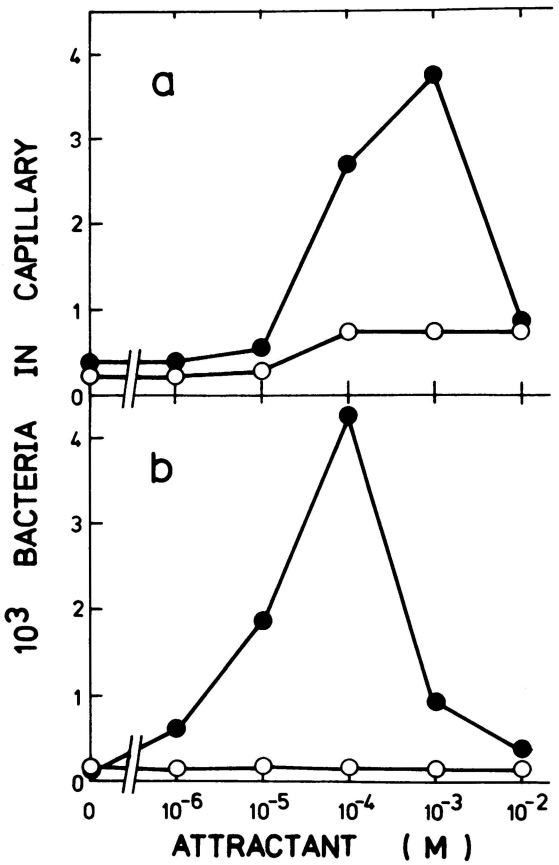

FIG. 4. Aspartate and maltose chemotaxis in *E. coli* MS5228 [(*tar_E-tap*)*5201 tsr_E*-1] with a plasmid carrying the *tar_E*$^+$ (pNM17) or the *tar_S*$^+$ (pRK41) gene. Capillary assays were performed at different concentrations of α-methyl-DL-aspartate (a) or maltose (b). Symbols: ●, MS5228(pNM17); ○, MS5228(pRK41).

transformants have the correct chemotransducers, as was expected.

Introduction of *S. typhimurium tar$_S$* gene into *E. coli* cells. For the control experiments, the transformants isolated by another combination of host and plasmid were prepared. Namely, either pNM17 or pRK41 was introduced into *E. coli* MS5228, which has defects in both Tar_E and Tsr_E proteins. The resultant transformants MS5228(pNM17) and MS5228(pRK41) were tested for aspartate and maltose chemotaxis and also for the changes in the methylation level of their chemotransducers after stimulation.

MS5228(pNM17) exhibited chemotaxis to α-methyl-DL-aspartate (Fig. 4a). However, in MS5228(pRK41) the chemotaxis was significant but small. This is probably due to the tumbly phenotype of this transformant and also to the slow adaptation to the stimuli, as reported previously (23), and is consistent with the fact that the strain showed almost no swarms on a tryptone soft agar plate (data not shown). MS5228(pNM17) showed normal maltose chemotaxis but MS5228(pRK41) did not (Fig. 4b).

The temporal stimulation assay was performed to confirm the results given above. Both transformants showed smooth swimming when 0.1 mM α-methyl-DL-aspartate was added, whereas the addition of 0.1 mM maltose caused smooth swimming only in MS5228(pNM17) (Table1).

The methylation of chemotransducers in MS5228 was almost negligible under our methylation conditions. The transformants MS5228(pNM17) and MS5228(pRK41) showed significant incorporation of radioactivity, indicating that the Tar_E and Tar_S proteins in these transformants are active substrates for the methylation reaction. By the addition of either α-methyl-DL-aspartate or maltose, Tar_E proteins in MS5228(pNM17) showed an increase in the methylation level (Fig. 5a). In the case of Tar_S proteins in MS5228(pRK41), however, the methylation level was increased by α-methyl-DL-aspartate but not by maltose (Fig. 5b). The results are consistent with the behavioral data of

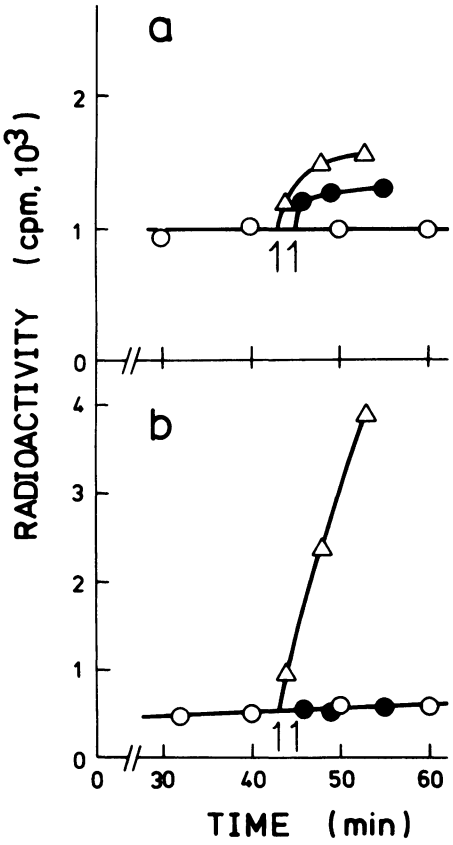

FIG. 5. Changes in the methylation level of Tar_E or Tar_S proteins in MS5228(pNM17) (a) and MS5228(pRK41) (b). The methylation level was measured as described in the legend to Fig. 2. At the arrows, cells received distilled water (○), 10 mM α-methyl-DL-aspartate (△), or 10 mM maltose (●).

these transformants. Furthermore, the fluorogram of Tar_E and Tar_S proteins of these transformants showed essentially the same migration pattern as shown in Fig. 3 (data not shown), indicating that the transformants had expected chemotransducers.

DISCUSSION

Unlike *E. coli*, *S. typhimurium* has no ability to show maltose chemotaxis. For maltose chemotaxis in *E. coli*, presence of maltose-binding protein in the periplasm and of a chemotransducer (Tar_E protein) for aspartate and maltose in the membrane is essential (9, 22). Therefore, the absence of maltose chemotaxis in *S. typhimurium* is considered to be caused by a defect in either maltose-binding protein or Tar_S protein, a *S. typhimurium* chemotransducer corresponding to Tar_E protein, or both. In this paper, we showed that *S. typhimurium* acquired maltose chemotaxis by the introduction of Tar_E proteins from *E. coli*, whereas the introduction of Tar_S proteins into a Tar_E-deficient *E. coli* restored only aspartate chemotaxis but not maltose chemotaxis. Thus, it is clear that Tar_S protein but not maltose-binding protein or membrane properties has the responsibility for the absence of maltose chemotaxis in *S. typhimurium*.

Recently, Dahl and Manson (6a) found that maltose-binding proteins isolated from *S. typhimurium* had an ability to restore maltose chemotaxis in a maltose-binding protein-deficient *E. coli*. Their results eliminated the possibility that maltose-binding protein in *S. typhimurium* is the cause of the absence of maltose chemotaxis in *S. typhimurium*. Their results are not only consistent but also complementary with ours.

In the capillary assay, we found that *S. typhimurium* cells with Tar_E protein showed significant efficiency in maltose chemotaxis and almost the same maltose concentration for peak accumulation of the cells compared with *E. coli* cells with Tar_E proteins. The results suggest that the maltose-binding protein of *S. typhimurium* can normally interact with a foreign chemotransducer, Tar_E protein, which was embedded in the membrane of *S. typhimurium*. Thus, the difference in membrane properties between *E. coli* and *S. typhimurium* for the function of the Tar protein might be small.

Both the Tar_E and Tar_S proteins are known to be the aspartate chemoreceptor-transducer, and the amino acid sequences of these proteins, which are estimated from DNA sequence data of corresponding genes, have a quite high similarity, about 78% in total amino acids (12, 23). However, our results show that Tar_E protein has sites for both aspartate and maltose-binding proteins but Tar_S protein has only a site for aspartate. Then, we compared the amino acid sequences of the periplasmic domain in these chemotransducers, because the sites for aspartate or maltose-binding protein are suggested to be located in this domain (11, 12, 23). Based on the predicted amino acid sequences on these chemotransducers (12, 23), homology of the periplasmic domain of these chemotransducers is calculated to be about 67%, while homology of the cytoplasmic domain of these proteins is greater than 80%. Thus, the difference between Tar_E and Tar_S proteins is suggested to be mainly located in the receptor domain of the proteins.

Parkinson et al. (21) reported that they have succeeded in isolating a new type of tar_E-deficient *E. coli* mutant which shows chemotaxis to maltose but not to aspartate. The result supports the idea that the sites for aspartate and maltose-binding protein in Tar_E protein are different. Then, Tar_S protein is considered to correspond to another type of defect in Tar protein; a site for aspartate is still active but a site for maltose-binding protein is inactive.

ACKNOWLEDGMENTS

We thank L. R. Bullas of Loma Linda University, D. E. Koshland, Jr., of the University of California at Berkeley, J. S. Parkinson of the University of Utah, and M. I. Simon of the California Institute of Technology for providing us with bacterial strains and plasmids. We also thank M. D. Manson for the generous communication of results before publication.

The work was supported in part by grant-in-aid 60115006 to Y.I. from the Ministry of Education, Science, and Culture of Japan.

LITERATURE CITED

1. **Adler, J.** 1973. A method for measuring chemotaxis and use of the method to determine optimum conditions for chemotaxis in *Escherichia coli*. J. Gen. Microbiol. **74**:77–91.
2. **Adler, J.** 1975. Chemotaxis in bacteria. Ann. Rev. Biochem. **44**:341–356.
3. **Adler, J., G. L. Hazelbauer, and M. M. Dahl.** 1973. Chemotaxis toward sugars in *Escherichia coli*. J. Bacteriol. **115**:824–847.
4. **Amman, E., J. Brosius, and M. Ptashne.** 1983. Vectors bearing a hybrid *trp-lac* promoter useful for regulated expression of cloned genes in *Escherichia coli*. Gene **25**:167–178.
5. **Aswad, D., and D. E. Koshland, Jr.** 1975. Isolation, characterization and complementation of *Salmonella typhimurium* chemotaxis mutants. J. Mol. Biol. **97**:225–235.
6. **Bullas, L. R., and J.-I. Ryu.** 1983. *Salmonella typhimurium* LT2

strains which are r^-m^+ for all three chromosomally located systems for DNA restriction and modification. J. Bacteriol. **156**:471–474.

6a. **Dahl, M. K., and M. D. Manson.** 1985. Interspecific reconstitution of maltose transport and chemotaxis in *Escherichia coli* with maltose-binding protein from various enteric bacteria. J. Bacteriol. **164**:1057–1063.

7. **DeFranco, A. L., J. S. Parkinson, and D. E. Koshland, Jr.** 1979. Functional homology of chemotaxis genes of *Escherichia coli* and *Salmonella typhimurium*. J. Bacteriol. **139**:107–114.

8. **Hedblom, M. L., and J. Adler.** 1980. Genetic and biochemical properties of *Escherichia coli* mutants with defects in serine chemotaxis. J. Bacteriol. **144**:1048–1060.

9. **Koiwai, O., and H. Hayashi.** 1979. Studies on bacterial chemotaxis. IV. Interaction of maltose receptor with a membrane-bound chemosensing component. J. Biochem. (Tokyo) **86**: 27–34.

10. **Koshland, D. E., Jr.** 1980. Bacterial chemotaxis as a model behavioral system. Raven Press, New York.

11. **Krikos, A., M. P. Conley, A. Boyd, H. C. Berg, and M. I. Simon.** 1985. Chimeric chemosensory transducers of *Escherichia coli*. Proc. Natl. Acad. Sci. USA **82**:1326–1330.

12. **Krikos, A., N. Mutoh, A. Boyd, and M. I. Simon.** 1983. Sensory transducers of E. coli are composed of discrete structural and functional domains. Cell **33**:615–622.

13. **Maniatis, T., E. F. Fritsch, and J. Sambrook.** 1982. Molecular cloning, a laboratory manual, p. 250–251, 368–369. Cold Spring Harbor Laboratory, Cold Spring Harbor, N.Y.

14. **Manson, M., W. Boos, P. J. Bassford, Jr., and B. A. Rasmussen.** 1985. Dependence of maltose transport and chemotaxis on the amount of maltose-binding protein. J. Biol. Chem. **260**: 9727–9733.

15. **Mizuno, T., and Y. Imae.** 1984. Conditional inversion of the thermoresponse in *Escherichia coli*. J. Bacteriol. **159**:360–367.

16. **Oosawa, K., and Y. Imae.** 1983. Glycerol and ethylene glycol: members of a new class of repellents of *Escherichia coli*. J. Bacteriol. **154**:104–112.

17. **Oosawa, K., and Y. Imae.** 1984. Demethylation of methyl-accepting chemotaxis proteins in *Escherichia coli* induced by the repellents glycerol and ethylene glycol. J. Bacteriol. **157**:576–581.

18. **Palva, E. T., P. Liljestroem, and S. Harayama.** 1981. Cosmid cloning and transposon mutagenesis in *Salmonella typhimurium* using phage vehicles. Mol. Gen. Genet. **181**:153–157.

19. **Parkinson, J. S., and G. L. Hazelbauer.** 1983. Bacterial chemotaxis: molecular genetics of sensory transduction and chemotactic gene expression, p. 293–310. *In* Gene function in prokaryotes. Cold Spring Harbor Laboratory, Cold Spring Harbor, N.Y.

20. **Parkinson, J. S., and P. T. Revello.** 1978. Sensory adaptation mutants of E. coli. Cell **15**:1221–1230.

21. **Parkinson, J. S., M. K. Slocum, A. M. Callahan, D. Sherris, and S. E. Houts.** 1983. Genetics of transmembrane signaling proteins in *E. coli*, p. 563–577. *In* H. Sund and C. Veeger (ed.), Mobility and recognition in cell biology. Walter de Gruyter and Co., Berlin.

22. **Richarme, G.** 1982. Interaction of the maltose-binding protein with membrane vesicles of *Escherichia coli*. J. Bacteriol. **149**:662–667.

23. **Russo, A. F., and D. E. Koshland, Jr.** 1983. Separation of signal transduction and adaptation functions of the aspartate receptor in bacterial sensing. Science **220**:1016–1020.

24. **Silverman, M., and M. Simon.** 1977. Chemotaxis in *Escherichia coli*: methylation of *che* gene products. Proc. Natl. Acad. Sci. USA **74**:3317–3321.

25. **Springer, M. S., M. F. Goy, and J. Adler.** 1977. Sensory transduction in *Escherichia coli*: two complementary pathways of information processing that involve methylated proteins. Proc. Natl. Acad. Sci. USA **74**:3312–3316.

26. **Wang, E. A., and D. E. Koshland, Jr.** 1980. Receptor structure in the bacterial sensing system. Proc. Natl. Acad. Sci. USA **77**:7157–7161.